# Chapter 28
# The Neurosurgeon

Dirk De Ridder

**Keypoints**

1. Neurosurgeons can contribute in a similar fashion to treatments of tinnitus as they currently do in pain treatment.
2. Neurosurgeons should collaborate with other clinicians and basic neuroscientists to help elucidate the pathophysiology of tinnitus.
3. Invasive neuromodulation can be helpful in selected forms of intractable tinnitus.
4. Different intracranial pathologies exist that can cause tinnitus amenable to surgical treatment, both of the non-pulsatile and of the pulsatile type.
5. Non-pulsatile tinnitus can be considered analogous to pain and results from changes in neural networks of the brain.
6. Pulsatile tinnitus is mostly related to anomalies of blood vessels in and around the brain.

**Key words** Tinnitus • Neurosurgeon • Tinnitus • Pulsatile • Non-pulsatile • Neurosurgery • Neuromodulation

**Abbreviations**

| | |
|---|---|
| CPA | Cerebellopontine angle |
| CSF | Cerebrospinal fluid |
| ENT | Ear nose and throat |
| Gy | Gray (unit of absorbed radiation) |
| MRI | Magnetic resonance imaging |
| TMS | Transcranial magnetic stimulation |

D. De Ridder (✉)
BRAI²N and Department of Neurosurgery,
TRI Tinnitus Clinic Antwerp, University of Antwerp,
Wilrijkstraat 10, 2650 Edegem, Belgium
e-mail: dirk.de.ridder@uza.be

## Introduction

Tinnitus has traditionally been a field belonging to ear nose and throat (ENT) surgeons, audiologists, and psychiatrists, except for some forms of pulsatile tinnitus, such as anomalies of the cerebral blood vessels, which have usually been treated by neurosurgeons.

Recently, both basic research [1] and clinical research [2, 3] have focused on the brain's involvement in the generation of tinnitus, opening the tinnitus field up to neurologists and neurosurgeons specialized in the field of tinnitus (see also Chap. 26).

Neurosurgeons treat patients with pain in an invasive way, and based on the analogy between some forms of pain and tinnitus [4–7], both of which can be considered deafferentation or phantom phenomena [8], the step to treating tinnitus for neurosurgeons is not as big as it looks at first sight (see Chap. 94).

A patient's referral to a neurosurgeon for pain relief was once considered bad news, because the choice of procedures was limited to the creation of lesions, offering significant risk and only modest success [9]. Neurosurgery used to be considered the "pursuit of the impossible by the irrepressible" [10]. In a similar way, the tinnitus field still considers the neurosurgeon a last resort, when everything else fails and the patient is suicidal or distressed by the tinnitus. Neurosurgical approaches to tinnitus are still too often described as "the half mad being operated upon by the mad" [10].

Advances in technology and an improved understanding of pain have helped to develop more effective procedures to such an extent that a recent textbook [11]

A.R. Møller et al. (eds.), *Textbook of Tinnitus*,
DOI 10.1007/978-1-60761-145-5_28, © Springer Science+Business Media, LLC 2011

discusses more than 30 types of procedures used in more than 18 major categories of pain [9]. However, as stated in the textbook, this does not mean that the neurosurgical procedures should always be the first line of treatment, as chances for pain relief are greatest when neurosurgery is but one piece of a comprehensive plan incorporating all possible treatment modalities [9].

Neurosurgeons treat the cause of pain (for example, disk surgery and microvascular decompressions) or use invasive neuromodulation when the cause of the pain is unknown or cannot be treated. In a similar way, the neurosurgeon should be involved in tinnitus treatment, dealing with the cause of the tinnitus, and by using neuromodulation to treat the symptoms.

There are indeed a series of pathologies that can cause tinnitus, either as their principal symptom or as one in a constellation of symptoms. Knowing the clinical course of the tinnitus in these pathologies is needed in order to be able to prognostically address these pathologies surgically. Some examples of specific diseases that often have tinnitus among their symptoms and that can be treated surgically are vestibular schwannoma, Arnold-Chiari malformations, arachnoid cysts, and others. Treatment of such diseases belongs to the classical repertoire of neurosurgery. However, neuromodulation through electrical (or magnetic) stimulation or lesioning, which are effective methods in treating disorders such as tinnitus and some forms of pain, also belong to the armamentarium of modern neurosurgery. Although neurosurgical procedures are traditionally the last resort in the battle against tinnitus, it is of interest for the tinnitus field to learn from neurosurgical pain management, which has brought relief to many patients with pain where other treatments have been ineffective. There are reasons to believe that neurosurgical treatment of tinnitus may evolve to become as widely used for treatment of tinnitus as it is now for treatment of pain.

However, brain surgeons should not limit themselves to developing new treatments for tinnitus based on analogy with pain. As a brain surgeon, one has a unique and unparalleled access to the brain, permitting recordings directly from the brain. It is important that they team up with basic neuroscientists to collaborate in order to gain as much valuable information as possible during the short window of direct brain access [12]. The power of intraoperative studies of brain function has a long history beginning with Penfield in the 1930s [13], extending to modern times where large parts of our understanding of the function of many systems of the human brain is based on intraoperative studies in patients undergoing neurosurgical operations [14, 15].

The neurosurgeon treating tinnitus should ideally work in a multidisciplinary team consisting of not only clinicians but also basic neuroscientists. Therefore, as long as no standardized neurosurgical treatments become available for tinnitus suppression, the neurosurgeon should not limit himself/herself to be a "sophisticated manual laborer" but should also be a "researcher" attempting to better understand the pathophysiology of tinnitus in order to develop new treatments for this elusive symptom (see Chaps. 21, 90, and 94).

## Neurosurgical Approaches to Tinnitus

Tinnitus can be divided into two entirely different entities: pulsatile and non-pulsatile tinnitus [16–18]. Pulsatile tinnitus is usually related to vascular anomalies or intracranial hyper- or hypotension and is not related to an abnormal function of the auditory system. Non-pulsatile tinnitus, on the other hand, is critically related to an abnormal function of the auditory system.

### *Non-Pulsatile Tinnitus*

Non-pulsatile tinnitus can be considered an auditory phantom phenomenon [8], resulting from auditory deprivation or deafferentation [1]. Any lesion along the auditory tract altering its normal function can cause non-pulsatile tinnitus. Ménière's disease, vestibular schwannoma, cerebellopontine angle (CPA) lesions, arachnoid cysts, microvascular compressions, Chiari malformation, and brain tumors are causes of non-pulsatile tinnitus that can be treated surgically.

If no cause for a patient's tinnitus can be found and thus no causal treatment can be offered, attempts to provide permanent relief from treatments such as electrical stimulation should be tried. First, non-invasive stimulations at different targets of the auditory system (promontory stimulation, transcranial magnetic stimulation

[TMS], transcranial direct current stimulation, transcutaneous electrical nerve stimulation) to test if a permanent implant could be beneficial should be performed. However, no prognostic relation has yet been shown between the effect of TMS aimed at the auditory cortex or cortical electrical stimulation at the level of the auditory cortex.

In *Vestibular schwannoma*, a high-pitch tinnitus (described as ringing or steam from a kettle) is present in 60–85% of the participants in a recent study [19]. Since the advent of stereotactic irradiation, vestibular schwannomas are often treated by radiosurgery, especially gamma knife radiosurgery. This seems to have a similar effect on tinnitus as microsurgery, although it seems to induce less tinnitus in the short term after treatment. Studies have shown that the tinnitus in 12–46% of such patients improves after the treatment [20] and tinnitus develops in only 4% of the patients after radiosurgery [20, 21]. The tinnitus experienced by patients who underwent microscopic surgery for removal of vestibular schwannoma disappeared in 16–50% of the participants in a study [22, 23]. Other studies have shown that after surgery to remove vestibular schwannoma, the tinnitus is reduced in 16% of patients, in 55% it does not change, and in 29% it becomes worse [22, 24, 25], especially when hearing is saved in surgery [24]. While the prevalence of tinnitus before and after operations, where hearing preservation is not attempted, is not significantly different, there are significant differences in tinnitus before and after operations where hearing is saved. When tinnitus is absent preoperatively, 85% of the hearing preservation group develops tinnitus after the operation while only 31% of patients in whom hearing preservation was not attempted developed tinnitus [24]. The results of other studies are, however, more optimistic, showing that only 8% of patients developed tinnitus after hearing preservation operations for vestibular schwannoma [26].

Gamma knife treatment has advantages over surgery as well as disadvantages. Gamma knife radiosurgery is less invasive and requires shorter hospitalization and convalescence periods [27]. The development of facial palsy or paresis is extremely rare (1% if irradiation dose is <14 Gy), and hearing can be saved in almost 80% of patients if 13 Gy as the maximum dose is respected [28]. The technique is, however, limited to lesions less than 3 cm in size and carries a greater risk for the development of post-treatment hydrocephalus and a certain, though small, risk of dedifferentiation into a neoplasia (malignancy). In small lesions (<1.5 cm) without serviceable hearing microsurgery and gamma knife treatment have comparable rates of tinnitus, tumor control, facial nerve function, and trigeminal function. However, stereotactic radiosurgery has a greater risk of long-term balance problems compared to microsurgery [29]. In general, gamma knife surgery might be better for vestibular schwannoma treatment in the short term [30], with similar effects on improvement and worsening of tinnitus as surgery. Surgery after failed gamma knife treatment has an increased risk for facial palsy due to strong adhesions [31] (see Chap. 85).

Other CPA *lesions* [32] such as meningioma, epidermoid tumors, lipoma, choroid plexus papilloma, epithelial cysts, teratoma, cavernoma, and hemangioma are sometimes associated with non-pulsatile tinnitus, usually together with other symptoms depending on the location of the lesion and the degree of brainstem, cerebellar, or cranial nerve compression.

*Arachnoid cysts* are a rare cause of non-pulsatile tinnitus. It is a congenital or posttraumatic/post-inflammatory disorder [33, 34], leading to vague symptoms [35]. However, infratentorial [36] arachnoid cysts can sometimes mimic Ménière's disease as well. Arachnoid cysts producing tinnitus can occur in the CPA [35, 37, 38], but also retroclival, retrocerebellar, and lateral of the cerebellum [39], with postoperative improvement of the tinnitus [39]. Usually, symptoms of intracranial hypertension are associated with non-pulsatile tinnitus [35, 40]. Surgical treatment consists of marsupialization[1] or excision of the cyst [40]. Also supratentorial Sylvian fissure arachnoid cysts can generate isolated tinnitus, and tinnitus suppression can be the result of marsupialization of the cysts if they act as a mass lesion [41]. Supratentorial cysts can also mimic Ménière's disease [42]. Imaging studies using intrathecal contrast to verify if an arachnoid cyst-like lesion communicates with normal cerebrospinal fluid (CSF) flow can help to ascertain whether an arachnoid cyst could act as a mass lesion and thus be symptomatic or not. Magnetic resonance imaging (MRI) sequences looking for a flow void within the cyst can be helpful as well [43].

[1] Marsupialization: Surgical alteration of a cyst or similar enclosed cavity by making an incision and suturing the flaps to the adjacent tissue, creating a pouch. (From: The American Heritage® Stedman's Medical Dictionary.)

*Ménière's syndrome* is a clinical entity consisting of episodic vertigo, fluctuating sensory hearing loss, tinnitus, and aural fullness (see Chap. 38). This syndrome is caused histopathologically by endolymphatic hydrops that can be caused by many pathologies – traumatic (acoustic, iatrogenic, or temporal bone trauma and labyrinthine concussion), infectious/inflammatory (autoimmune inner ear, see Chap. 60), Cogan's syndrome, chronic otitis media, viral or serous labyrinthitis, syphilis, tumoral (leukemia), congenital (deafness, Mondini dysplasia), or in the setting of connective tissue or bone disease (Letterer-Siwe disease, Paget's disease, otosclerosis), and others [44]. Tinnitus worsens both in intensity and as a function of duration and bilateral disease [45]. It is perceived as worse than in a comparable group of tinnitus sufferers due to acoustic trauma or otosclerosis [45].

In Ménière's disease, any kind of surgery, whether vestibular nerve section, cochlear nerve section, endolymphatic sac surgery [46], or gentamicin injections [47], never seems to produce greater than 50% tinnitus control – a small improvement upon the 30% spontaneous disappearance in its natural history [48]. Endolymphatic sac surgery, independent on whether decompression, exclusion, or shunting is done, improves or cures tinnitus in 40% of patients with Ménière's disease [49]. This is similar to intratympanic gentamycin application, a less invasive technique with 27–69% tinnitus improvement [50–52].

In a recent review paper on vestibular nerve section performed for tinnitus [53], the proportion of patients in whom tinnitus was exacerbated postoperatively ranged from 0 to 60%, with a mean of 16.4%. The proportion of patients in whom tinnitus was unchanged was 17–72% (mean 38.5%), and in whom tinnitus was improved was 6–61% (mean 37.2%). These results are similar to gentamycin and endolymphatic sac surgery. In the majority of patients undergoing vestibular nerve section, ablation of auditory efferent input (and thus total efferent dysfunction) to the cochlea was not associated with an exacerbation of tinnitus [53].

In *otosclerosis*, tinnitus is very common; up to 91% of individuals with otosclerosis have tinnitus and 38% is severely affected by it [54]. Successful stapedectomy causes disappearance of non-pulsatile tinnitus in up to 40–73% tinnitus [55–60] with another 32–37% improving. In individuals who did not have tinnitus before stapedectomy, the risk of developing it after the surgery is almost non-existent. Only in 10%, the operation does not improve the tinnitus [60] and in another 8% it worsens [57]. Rarely, otosclerosis also produces arterial pulsatile tinnitus, due to a neovascularization at the site of stapes fusion. Stapedectomy can sometimes cure this rare form of pulsatile tinnitus [17].

A *tumor* in the auditory cortex, compressing the auditory cortex, can cause ipsilateral fluctuating non-pulsatile tinnitus as the sole symptom, probably due to a direct influence on normal cortical sound processing. Removal of the lesion resulted in abolishing the tinnitus in 4 out of 5 patients who had the operation [41]. Tumors elsewhere along the auditory tract (for example, the brainstem) rarely present with tinnitus only but usually give rise to additional symptoms related to the tumor's closeness of other neural structures in the brainstem.

For *intractable non-pulsatile tinnitus*, auditory brainstem implants [61] (see Chap. 77) and auditory cortex stimulations can give relief in intractable non-pulsatile tinnitus [62–64]. These treatments are based on a recently developed pathophysiological model for non-pulsatile tinnitus, based on auditory deprivation or deafferentation as the initial trigger for tinnitus generation. Studies have shown that a decrease of auditory input induces a slowing of auditory information processed in the thalamocortical loop generating slow wave activity (delta en theta oscillations) [7, 65], with a decrease in lateral inhibition [66] and a halo or edge of increased activity [7, 67]. This is also called thalamocortical dysrhythmia [7] associated with cortical reorganization [68, 69]. The most likely mechanism that links hyperactivity and reorganization is synchrony [1]. Synchronization of the gamma band activity could possibly induce topographical reorganization via simple Hebbian mechanisms (cells that fire together wire together) [1]. Therefore, it seems logical to try and modify this tinnitus-related auditory cortex reorganization/hyperactivity in an attempt to suppress the tinnitus. This can be achieved using neuronavigation-guided TMS, a technique that is capable of modulating cortical activity. If TMS is capable of suppressing tinnitus, the effect could be maintained by implantation of electrodes at the area of signal abnormality on the auditory cortex. The first results in

patients with unilateral pure-tone tinnitus have shown statistically significant tinnitus suppression, without suppressing white or narrow band noise in individuals who responded to TMS with decreased tinnitus [63]. More recent trials also suggested that narrow band tinnitus is suppressible with novel stimulation designs consisting of closely spaced spikes of very high frequencies [70].

Reafferentation of the auditory thalamocortical system after it has been deprived of input can also be achieved by cochlear implants (see Chap. 77). Almost, immediately after the introduction of cochlear implants for hearing improvement, it was noted that the electrical intracochlear stimulation ameliorated tinnitus in a large proportion of individuals [71, 72]. Multiple studies since then have replicated these results indicating that cochlear implants inserted for hearing improvement can also modulate tinnitus [73–77], not only unilaterally but also bilaterally in a majority of individuals [78]. A recent study using cochlear implant insertion in patients with incapacitating tinnitus and ipsilateral complete hearing loss and contralateral preserved hearing demonstrates similarly promising results [79]. Using promontory stimulation as a preoperative non-invasive test in this selected group of patients might predict good outcomes in tinnitus suppression.

A limit to this technique is that it can only be used in patients with unilateral complete hearing loss. This could potentially be extended to people with high-frequency hearing loss but preserved low-frequency hearing, as a recent paper has shown that short hybrid cochlear implants can preserve low-frequency hearing [80]. Another option is to use extracochlear stimulation for tinnitus suppression. The first attempts for developing extracochlear electrical stimulation have been made [76, 81] as well.

### *Pulsatile and Pseudopulsatile Tinnitus*

Many causes of pulsatile tinnitus are amenable to interventional neuroradiological procedures or neurosurgical interventions, whereas most problems involving pseudopulsatile tinnitus are the domain of the ENT surgeon (see Table 28.1). For an overview of these pathologies, the reader is referred to Chap. 59.

**Table 28.1** Surgically treatable causes of tinnitus

| Pulsatile tinnitus | Non-pulsatile tinnitus |
|---|---|
| *Venous* | Vestibular schwannoma (acoustic neuroma) |
| Benign intracranial hypertension | Other cerebellopontine angle lesions |
| Chiari malformation | Arachnoid cyst |
| High jugular bulb | Menière's disease |
| Sigmoid sinus diverticulum | Otosclerosis |
| Sigmoid-transverse aneurysm | Microvascular compression |
| Aberrant veins aneursm | Chiari malformation |
| *Arterial* | Brain tumor |
| Carotid stenosis | *Symptomatic* |
| Aberrant carotid artery | Cochlear implant |
| Glomus tumor | Brainstem implant |
| Vascular lesions of petrous bone/skull base | Auditory cortex implant |
| Arteriovenous malformation | *Pseudopulsatile tinnitus* |
| Aneurysm | Palatal myoclonus |
| Canal dehiscence | Middle ear myoclonus |
| Benign intracranial hypertension | Patulous eustachian tube |
| Carotid-cavernous fistula | |
| Intrameatal vascular loop | |
| Somatosensory pulsatile tinnitus syndrome | |
| Idiopathic | |

## Conclusion

Stimulated by recent developments in our understanding of the pathophysiology of tinnitus, treatment has shifted from purely otological approaches to brain-based approaches. Therefore, neurosurgeons should become more involved in treating this elusive symptom.

Tinnitus actually consists of two entirely different entities with a different pathophysiology, different clinical symptoms, and different treatment. Before tinnitus patients are told "to learn to live with their tinnitus" it can be suggested to look for possible causes for both non-pulsatile and pulsatile tinnitus as this can result in an otoneurosurgical treatment. In patients with non-pulsatile tinnitus, non-invasive trials with promontory or TMS can potentially help select candidates for a permanent implant as a treatment for tinnitus. Neurosurgeons should be involved not only in the surgical treatment of operable causes but also in the exploration of possible pathophysiological mechanisms, making use of their unique ability to

record activity directly from the brain when performing intracranial surgery.

## References


1. Eggermont JJ and LE Roberts (2004) The neuroscience of tinnitus. Trends Neurosci 27:676–82.
2. Lockwood AH, RJ Salvi and RF Burkard (2002) Tinnitus. N Engl J Med 347:904–10.
3. Lockwood AH, RJ Salvi, RF Burkard et al (1999) Neuroanatomy of tinnitus. Scand Audiol Suppl 51:47–52.
4. Møller AR (2000) Similarities between severe tinnitus and chronic pain. J Am Acad Audiol 11:115–24.
5. Møller AR (1997) Similarities between chronic pain and tinnitus. Am J Otol 18:577–85.
6. Tonndorf J (1987) The analogy between tinnitus and pain: a suggestion for a physiological basis of chronic tinnitus. Hear Res 28:271–5.
7. Llinas RR, U Ribary, D Jeanmonod et al (1999) Thalamocortical dysrhythmia: a neurological and neuropsychiatric syndrome characterized by magnetoencephalography. Proc Natl Acad Sci USA 96:15222–7.
8. Jastreboff PJ (1990) Phantom auditory perception (tinnitus): mechanisms of generation and perception. Neurosci Res 8: 221–54.
9. Giller CA (2003) The neurosurgical treatment of pain. Arch Neurol 60:1537–40.
10. Dan NG (1995) The personality of the neurosurgeon. Acta Neurochir (Wien) 132:217–21.
11. Burchiel KJ (ed) (2002) Surgical management of pain. Thieme: New York.
12. Abbott A (2009) Neuroscience: Opening up brain surgery. Nature 461:866–8.
13. Penfield W and E Boldrey (1937) Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. Brain 60:389–443.
14. Anderson WS, SO O'Hara, HC Lawson et al (2006) Plasticity of pain-related neuronal activity in the human thalamus. In Reprogamming the brain, AR Møller (Ed). Elsevier: Amsterdam, 353–64.
15. Ojemann GA (2003) The neurobiology of language and verbal memory: observations from awake neurosurgery. Int J Psychophysiol 48:141–6.
16. Sismanis A (1997) Pulsatile tinnitus. Int Tinnitus J 3: 39–40.
17. Sismanis A (1998) Pulsatile tinnitus. A 15-year experience. Am J Otol 19:472–7.
18. Sismanis A (2003) Pulsatile tinnitus. Otolaryngol Clin North Am 36:389–402, viii.
19. Kentala E and I Pyykko (2001) Clinical picture of vestibular schwannoma. Auris Nasus Larynx 28:15–22.
20. Selch MT, A Pedroso, SP Lee et al (2004) Stereotactic radiotherapy for the treatment of acoustic neuromas. J Neurosurg 101 Suppl 3:362–72.
21. Hempel JM, E Hempel, B Wowra et al (2006) Functional outcome after gamma knife treatment in vestibular schwannoma. Eur Arch Otorhinolaryngol 263:714–8.
22. Fahy C, TP Nikolopoulos and GM O'Donoghue (2002) Acoustic neuroma surgery and tinnitus. Eur Arch Otorhinolaryngol 259:299–301.
23. Dandy W (1941) Surgical treatment of Ménière's disease. Surg Gynaecol Obstet 72:421–5.
24. Kanzaki J, A Satoh and T Kunihiro (1999) Does hearing preservation surgery for acoustic neuromas affect tinnitus? Skull Base Surg 9:169–76.
25. Berliner KI, C Shelton, WE Hitselberger et al (1992) Acoustic tumors: effect of surgical removal on tinnitus. Am J Otol 13:13–7.
26. Catalano PJ and KD Post (1996) Elimination of tinnitus following hearing preservation surgery for acoustic neuromas. Am J Otol 17:443–5.
27. Karpinos M, BS Teh, O Zeck et al (2002) Treatment of acoustic neuroma: stereotactic radiosurgery vs. microsurgery. Int J Radiat Oncol Biol Phys 54:1410–21.
28. Regis J, M Tamura, C Delsanti et al (2008) Hearing preservation in patients with unilateral vestibular schwannoma after gamma knife surgery. Prog Neurol Surg 21:142–51.
29. Coelho DH, JT Roland, Jr., SA Rush et al (2008) Small vestibular schwannomas with no hearing: comparison of functional outcomes in stereotactic radiosurgery and microsurgery. Laryngoscope 118:1909–16.
30. Pollock BE (2008) Vestibular schwannoma management: an evidence-based comparison of stereotactic radiosurgery and microsurgical resection. Prog Neurol Surg 21:222–7.
31. Shuto T, S Inomori, S Matsunaga et al (2008) Microsurgery for vestibular schwannoma after gamma knife radiosurgery. Acta Neurochir (Wien) 150:229–34; discussion 34.
32. Lalwani AK (1992) Meningiomas, epidermoids, and other nonacoustic tumors of the cerebellopontine angle. Otolaryngol Clin North Am 25:707–28.
33. Yanaka K, T Enomoto, T Nose et al (1988) Post-inflammatory arachnoid cyst of the quadrigeminal cistern. Observation of development of the cyst. Childs Nerv Syst 4:302–5.
34. Lesoin F, P Dhellemmes, M Rousseaux et al (1983) Arachnoid cysts and head injury. Acta Neurochir (Wien) 69:43–51.
35. Hadley MN, TW Grahm, CP Daspit et al (1985) Otolaryngologic manifestations of posterior fossa arachnoid cysts. Laryngoscope 95:678–81.
36. O'Reilly RC and EK Hallinan (2003) Posterior fossa arachnoid cysts can mimic Meniere's disease. Am J Otolaryngol 24:420–5.
37. Takano S, T Maruno, S Shirai et al (1998) Facial spasm and paroxysmal tinnitus associated with an arachnoid cyst of the cerebellopontine angle – case report. Neurol Med Chir (Tokyo) 38:100–3.
38. Zennaro O, F San-Gali, V Darrouzet et al (1992) Arachnoid cysts of the cerebellopontine angle. Rev Laryngol Otol Rhinol (Bord) 113:47–50.
39. Ottaviani F, CB Neglia, A Scotti et al (2002) Arachnoid cyst of the cranial posterior fossa causing sensorineural hearing loss and tinnitus: a case report. Eur Arch Otorhinolaryngol 259:306–8.
40. Haberkamp TJ, EM Monsell, WF House et al (1990) Diagnosis and treatment of arachnoid cysts of the posterior fossa. Otolaryngol Head Neck Surg 103:610–4.
41. Kovacs S, R Peeters, M Smits et al (2006) Activation of cortical and subcortical auditory structures at 3 T by means of a

functional magnetic resonance imaging paradigm suitable for clinical use. Invest Radiol 41:87–96.
42. Buongiorno G and G Ricca (2003) Supratentorial arachnoid cyst mimicking a Meniere's disease attack. J Laryngol Otol 117:728–30.
43. De Ridder D, G Alessi, M Lemmerling et al (2002) Hemilingual spasm: a new neurosurgical entity? Case report. J Neurosurg 97:205–7.
44. Borjab DI, SA Bhansali and RA Battista (1994) Peripheral vestibular disorders. In Neurotology, R Jackler, D Brackmann (eds). Mosby: St. Louis.
45. Herraiz C, MC Tapia and G Plaza (2006) Tinnitus and Meniere's disease: characteristics and prognosis in a tinnitus clinic sample. Eur Arch Otorhinolaryngol 263:504–9.
46. Meyershoff W (1992) Tinnitus. In Otolaryngology head and neck surgery, W Meyershoff, D Ria (Eds). Saunders Company: Philadelphia, 435–46.
47. Kaasinen S, I Pyykko, H Ishizaki et al (1995) Effect of intratympanically administered gentamicin on hearing and tinnitus in Meniere's disease. Acta Otolaryngol Suppl 520 Pt 1:184–5.
48. Vernon J, R Johnson and A Schleuning (1980) The characteristics and natural history of tinnitus in Meniere's disease. Otolaryngol Clin North Am 13:611–9.
49. Quaranta N, G De Thomasis, A Gandolfi et al (2001) Long-term audition results in patients with chronic endolymphatic hydrops after selective vestibular neurotomy and endolymphatic sac surgery. Acta Otorhinolaryngol Ital 21:131–7.
50. Quaranta A, A Scaringi, A Aloidi et al (2001) Intratympanic therapy for Meniere's disease: effect of administration of low concentration of gentamicin. Acta Otolaryngol 121:387–92.
51. Seidman M (2002) Continuous gentamicin therapy using an IntraEAR microcatheter for Meniere's disease: a retrospective study. Otolaryngol Head Neck Surg 126:244–56.
52. De Valck CF, V Van Rompaey, EL Wuyts et al (2009) Tenotomy of the tensor tympani and stapedius tendons in Meniere's disease. B-Ent 5:1–6.
53. Baguley DM, P Axon, IM Winter et al (2002) The effect of vestibular nerve section upon tinnitus. Clin Otolaryngol Allied Sci 27:219–26.
54. Sobrinho PG, CA Oliveira and AR Venosa (2004) Long-term follow-up of tinnitus in patients with otosclerosis after stapes surgery. Int Tinnitus J 10:197–201.
55. Glasgold A and F Altmann (1966) The effect of stapes surgery on tinnitus in otosclerosis. Laryngoscope 76:1524–32.
56. Gersdorff M, J Nouwen, C Gilain et al (2000) Tinnitus and otosclerosis. Eur Arch Otorhinolaryngol 257:314–6.
57. Ayache D, F Earally and P Elbaz (2003) Characteristics and postoperative course of tinnitus in otosclerosis. Otol Neurotol 24:48–51.
58. Oliveira CA (2007) How does stapes surgery influence severe disabling tinnitus in otosclerosis patients? Adv Otorhinolaryngol 65:343–7.
59. Sparano A, JP Leonetti, S Marzo et al (2004) Effects of stapedectomy on tinnitus in patients with otosclerosis. Int Tinnitus J 10:73–7.
60. Szymanski M, W Golabek and R Mills (2003) Effect of stapedectomy on subjective tinnitus. J Laryngol Otol 117:261–4.
61. Soussi T and SR Otto (1994) Effects of electrical brainstem stimulation on tinnitus. Acta Otolaryngol 114:135–40.
62. De Ridder D, G De Mulder, V Walsh et al (2004) Magnetic and electrical stimulation of the auditory cortex for intractable tinnitus. Case report. J Neurosurg 100:560–4.
63. De Ridder D, G De Mulder, E Verstraeten et al (2006) Primary and secondary auditory cortex stimulation for intractable tinnitus. ORL J Otorhinolaryngol Relat Spec 68:48–54; discussion 55.
64. De Ridder D, G De Mulder, E Verstraeten et al (2007) Auditory cortex stimulation for tinnitus. Acta Neurochir Suppl 97:451–62.
65. Weisz N, S Moratti, M Meinzer et al (2005) Tinnitus perception and distress is related to abnormal spontaneous brain activity as measured by magnetoencephalography. PLoS Med 2:e153.
66. Llinas R, FJ Urbano, E Leznik et al (2005) Rhythmic and dysrhythmic thalamocortical dynamics: GABA systems and the edge effect. Trends Neurosci 28:325–33.
67. Weisz N, S Muller, W Schlee et al (2007) The neural code of auditory phantom perception. J Neurosci 27:1479–84.
68. Muhlnickel W, T Elbert, E Taub et al (1998) Reorganization of auditory cortex in tinnitus. Proc Natl Acad Sci USA 95:10340–3.
69. Norena AJ and JJ Eggermont (2006) Enriched acoustic environment after noise trauma abolishes neural signs of tinnitus. Neuroreport 17:559–63.
70. De Ridder D, S Vanneste, E van der Loo et al (2010) Burst stimulation of the auditory cortex: a new form of neurostimulation for noise-like tinnitus suppression. J Neurosurg 112:1289–94.
71. Brackmann DE (1981) Reduction of tinnitus in cochlear-implant patients. J Laryngol Otol Suppl 4:163–5.
72. House JW and DE Brackmann (1981) Tinnitus: surgical treatment. Ciba Found Symp 85:204–16.
73. Miyamoto RT, MK Wynne, C McKnight et al (1997) Electrical suppression of tinnitus via cochlear implants. Int Tinnitus J 3:35–8.
74. Aschendorff A, G Pabst, T Klenzner et al (1998) Tinnitus in cochlear implant users: the Freiburg experience. Int Tinnitus J 4:162–4.
75. Di Nardo W, I Cantore, F Cianfrone et al (2007) Tinnitus modifications after cochlear implantation. Eur Arch Otorhinolaryngol 264:1145–9.
76. Hazell JW, LJ Meerton and MJ Conway (1989) Electrical tinnitus suppression (ETS) with a single channel cochlear implant. J Laryngol Otol Suppl 18:39–44.
77. Ito J and J Sakakihara (1994) Tinnitus suppression by electrical stimulation of the cochlear wall and by cochlear implantation. Laryngoscope 104:752–4.
78. Yonehara E, R Mezzalira, PR Porto et al (2006) Can cochlear implants decrease tinnitus? Int Tinnitus J 12:172–4.
79. Van de Heyning P, K Vermeire, M Diebl et al (2008) Incapacitating unilateral tinnitus in single-sided deafness treated by cochlear implantation. Ann Otol Rhinol Laryngol 117:645–52.
80. Gantz BJ, C Turner, KE Gfeller et al (2005) Preservation of hearing in cochlear implant surgery: advantages of combined electrical and acoustical speech processing. Laryngoscope 115:796–802.
81. Matsushima J, N Sakai, M Sakajiri et al (1996) An experience of the usage of electrical tinnitus suppressor. Artif Organs 20:955–8.